CLINICAL PROCEEDINGS

of the

CHILDREN'S HOSPITAL

WASHINGTON, D. C.

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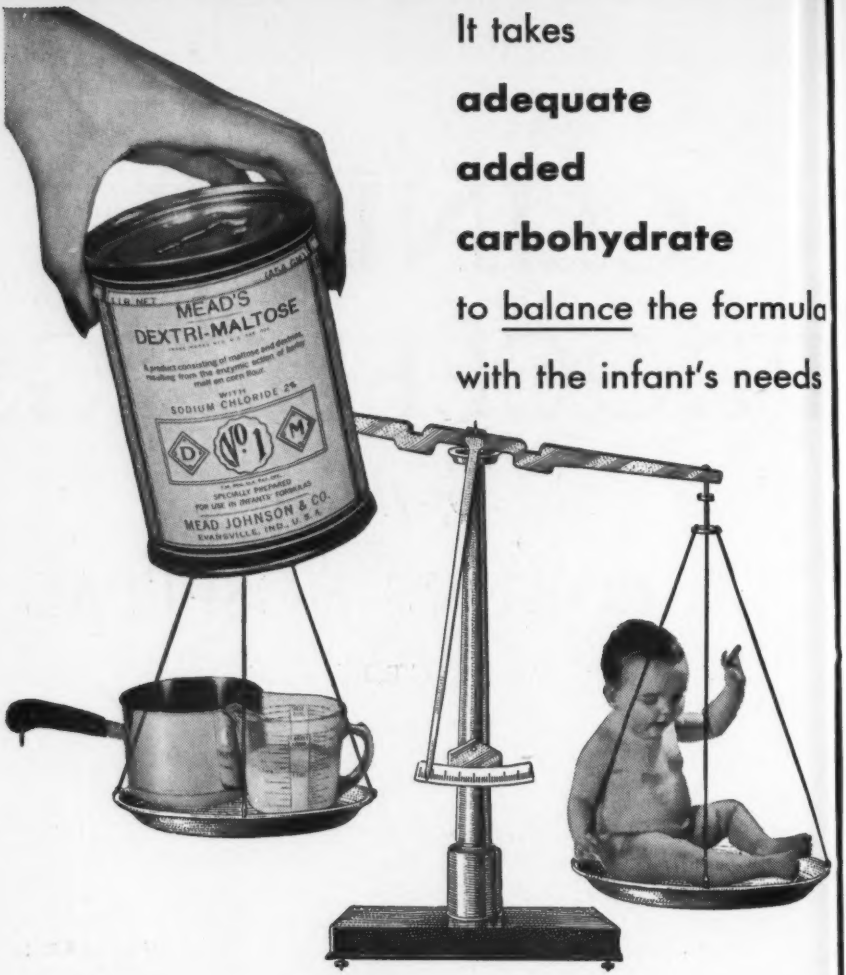

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CLINICAL PROCEEDINGS

OF THE CHILDRENS HOSPITAL

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1. Wetzel, N. C. et al.: Science 110:651, 1949.

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SULFONAMIDE REACTIONS

Editorial

Certain medical facts are so well known that they are too often overlooked. The purpose of this discussion is to re-emphasize the possible toxic effects which may accrue from the therapeutic administration of sulfonamide. There have appeared in the literature numerous instances of toxic reactions attributable to this drug. Despite the addition to the therapeutic medical armamentarium of newer, less toxic, and possibly more efficacious antibiotics, sulfonamide still enjoys widespread use, particularly among pediatricians. The ease of oral administration of the drug, its low cost, and ready availability are the factors responsible for such continued popularity. Most of the toxic reactions are of a mild and transitory nature and quickly subside when administration of the drug is discontinued, but occasionally, they are followed by death. It is not the intention of this editorial to overemphasize the dangers of this extremely valuable drug, but by re-emphasizing such reactions, it is our purpose to point out clinically observed complications and their management. In addition, stress is placed on the physician's responsibility to exercise caution in administering such a desirable, albeit, potentially dangerous drug.

The overall incidence of sulfonamide sensitivity is approximately 5 per cent. However, in the pediatric group such toxic reactions are not only less frequent but also less severe, occurring in approximately 1.5 per cent. The renal complications, the most frequent causes of fatalities, occur in less than 1 per cent of the pediatric patients treated with this drug.

Untoward reactions, which may occur as a result of sulfonamide therapy, may be classified according to the systems in which clinical symptoms of toxicity are manifested: the kidneys, blood and blood-forming organs, gastro intestinal tract, and central nervous system. Additional effects sometimes are produced which may be classified as systemic.

Kidneys

Renal symptoms are the most commonly encountered. They comprise over 50 per cent of the sulfonamide reactions and include hematuria, oliguria and anuria. Two main pathogenetic actions may produce renal complications. The first is a mechanical one. In this type crystals of the insoluble acetylated form of sulfonamide cause bleeding by irritation of the renal endothelium, or the accumulated crystals plug the tubules or ureters. In the second, which has been termed toxic, the drug produces effects on the renal parenchyma which result in areas of focal necrosis and/or degenerative changes in the renal tubules. Similar necrotic changes may occur in

other organs. The presence in the urine of sulfonamide crystals alone is *not* a reason to discontinue the use of the drug. The aims in the treatment of these renal complications are directed toward insuring an adequate urinary flow and reducing the concentration of the drug in the urine.

1. Hematuria: This manifestation usually occurs when large dosages are employed and is seen most frequently on the second or third day of treatment. Should microscopic hematuria be the only finding, the drug is discontinued for one or two days, or the dosage is reduced and the fluid intake increased by oral or parenteral routes. When microscopic hematuria continues or gross hematuria is encountered, the administration of the drug is discontinued immediately and parenteral fluids are given. In gross hematuria beneficial results have been reported with large parenteral dosages of ascorbic acd. If it becomes necessary to resume therapy, smaller dosages are recommended along with large amounts of fluid.

2. Oliguria or anuria: These complications are the most common causes of death and when they are present, it is mandatory to cease administration of the drug immediately. In an occasional case it will be necessary to resort to ureteral catheterization. It is also recommended that $\frac{1}{6}$ molar lactate be administered intravenously in an attempt to alkalinize the urine and render the crystals more soluble.

Blood and Blood-Forming Organs

Reactions of this nature are seen at the end of the first week or during the second week of treatment. They are manifested by the development of leukopenia, hemolytic anemia, jaundice, or purpura. When such complications arise during the course of treatment, they are frequently associated with other evidence of toxicity, e.g., fever and rash.

1. Leukopenia: Leukocyte counts below 5,000 white blood cells per cubic milliliter are seen in 2 to 3 per cent or patients treated with sulfonamide. If the white count is between 4,000 and 5,000 per cubic milliliter, treatment is not discontinued. Daily counts of the white blood cells are made and, in the majority, the number of cells will gradually return to normal. Should the leukopenia be found to be progressive, then therapeutic measures are instituted. These measures include cessation of drug therapy along with blood transfusions.

2. Hemolytic anemia, jaundice, or purpura: These complications are rarely encountered during the course of sulfonamide treatment. An occasional patient with one of these complications has been reported in the medical literature. These allergic manifestations usually are not fatal, and respond to the administration of blood plus discontinuation of sulfonamide therapy.

Gastro-Intestinal Tract

Nausea and vomiting may become manifest on the first or second day of treatment. It is difficult to indict the sulfonamide as a cause of these symptoms since they occur with such frequency in many acute illnesses of childhood. It will be found that nausea and vomiting will disappear despite the continuation of therapy as the illness is brought under control. Should vomiting preclude the use of the oral route, the soluble sodium salt can be given parenterally.

Central Nervous System

Mental disturbances may occur on the first or second day of sulfonamide administration. Again it is difficult to assess the role of the drug in the production of these disturbances. They will clear with improvement in the patient's illness. When such complications arise, it is not necessary to stop the administration of the sulfonamide.

Systemic Effects

Fever and/or rash may occur during an initial course of therapy with sulfonamide or on repeated administration of the drug. These systemic symptoms occur alone or in combination with any of the preceding complications.

1. Initial administration: Fever with or without rash may become present on the fifth to the ninth day of an initial course of treatment with sulfonamide. The incidence has been estimated to be less than 1 per cent. These symptoms persist until the drug is discontinued. In over 50 per cent of the patients who develop fever, an associated rash will appear. This rash will assume either a scarletinoform or morbilliform nature. It is not unusual in such patients to find a marked leukocytosis with white cell counts over 70,000 per cubic milliliter of which over 90 per cent will be polymorphonucelar cells.

2. Repeated administration: A "febrile" reaction and exanthem will occur within the first 24 to 48 hours when sulfonamide is given to a patient who has previously demonstrated sensitivity. A second course of treatment is not more toxic than the first. If a patient has had an initial reaction to the drug, he is apt to have developed sensitivity and may react to a subsequent course with fever and rash. These manifestations almost always occur within the first 48 hours and have been termed the "immediate" or hypersensitivity reactions. Over one-half of the patients showing an initial "febrile" response are coincidentally sensitized to the drug. In such children, a different sulfonamide is less likely to produce allergic symptoms. In any event, a test dose of 0.25 grams of sulfonamide will usually reveal the pres-

ence of sensitivity. If cross-sensitization is present, it may be necessary to utilize one of the antibiotics.

In conclusion, sulfonamide sensitivity is infrequent in infants and children. An occasional fatality, usually due to renal involvement, has been reported. When a "febrile" reaction has occurred previously, the danger of a severe reaction exists on subsequent administration. There is no need to fear an increase in harmful effects from multiple courses than from the first course of therapy. Because of the low incidence of reactions, the recommendation is made that sulfonamide therapy be not withheld from infants and children because of fear of an untoward response. A knowledge of the harmful effects which may accrue and the management of complications which may arise is all that is necessary to render the physician cautious in prescribing this valuable drug.

J. M. L.

ACUTE CARDIAC ARREST

Special Report No. 214

Marshall C. Sanford, M.D.

Of all emergencies encountered in medicine, acute cardiac arrest is the most exigent. Until recently, it was popular opinion that nothing other than cardiac stimulants could be given or was even warranted in this condition. The prognosis is favorable in the majority of instances, however, if the condition is recognized immediately and steps are taken to institute both artificial respiration and artificial circulation. Moreover, no other reversible condition requires such prompt action to insure complete recovery, since the time between cessation of cardiac function and the production of irreparable brain damage is considered to be approximately four minutes.

Acute cardiac arrest most commonly occurs during the course of a surgical procedure and gives no warning of its approach. It has been reported in almost all types of operations and with any anesthetic agent or combination of anesthetics. Not all cases occur in the operating room, however; any agent which interferes with adequate oxygenation or circulation may precipitate this condition. Examples of this are: drowning, electric shock, heart disease, poisoning, or direct injury to the heart.

The etiology of acute cardiac arrest is variable, but it is well established that a period of anoxia usually precedes the cessation of cardiac function, whatever the cause. This anoxia may be caused by a direct effect of the anesthetic agent, by a paralysis of the respiratory muscles as with spinal an-

esthesia or excessive curarization; by a multiplicity of drugs; or by improper aeration of the lungs as with pneumonia, drowning, asphyxia, pneumothorax, or croup.

With regard to the so-called "vagovagal reflex," Sloan's[9] studies suggest that "vagal reflexes arising during intrathoracic operations are of little importance in well oxygenated patients. They may, however, play a part in augmenting an existing oxygen deficiency and may aid in producing cardiac arrest under conditions of severe hypoxia or asphyxia."

Anoxia damages all organs and structures, but the brain and myocardium are the most sensitive. The cells of the cerebral cortex are damaged before the respiratory center receives injury. Early correction of cardiac arrest is followed by recovery of a normal individual. If treatment is delayed, the surviving patient may sustain permanent brain damage or the damage may be so severe that death will result. The blood not only must be oxygenated but must be circulated if damage to the vital centers is to be avoided. Therefore, inadequate filling of the heart is a causative factor and may result from hemorrhage, cardiac failure, or direct injury to the heart.

It is the anesthetist who initially detects the cessation of the heart beat unless the operation is intrathoracic and the heart is exposed. As soon as the absence of pulse and blood pressure is discovered, the anesthetist notifies the surgeon to check for any nearby arterial pulsations. Time is precious and none should be lost in prolonged indecision and useless checking. If the patient suddenly becomes pallid and there is absence of palpable and visible vascular pulsations, it is safe to assume that the heart has stopped beating and one should institute treatment immediately.

It is unnecessary to state that the availability of cardiac stimulants and a few surgical instruments is mandatory, for improvising may cause disastrous delay. In the few minutes during which the damage is reversible, many things must be accomplished. One must have considered in advance the steps he would take if such an emergency arose. It requires considerable courage to open the chest and begin cardiac massage when the surgeon is performing a simple operation elsewhere in the body. He must overcome understandable mental inhibitions.

If resuscitative measures are not in progress within three minutes, the recovery of a normal individual is unlikely.[3] The operating room of every hospital should have a routine procedure which can be instituted without delay or confusion. Each member of the surgical team should know exactly what he is expected to accomplish in the valuable moments available to save the patient's life. Sterile technique is of secondary importance and time should not be lost in obtaining it. The surgeon's prime concern is to start the heart beating again. The anesthetist should establish an adequate airway and maintain artificial respiration with 100 per cent oxygen. All oper-

ating suites should have in readiness a cardiac kit containing a few basic instruments (scalpel, hemostats, ligatures, and retractors), and separately-wrapped syringes filled with cardiac stimulants; since even in the operating room the necessary adjuncts may not be available because the operation may not have started or may be already completed. An electrical defibrillator should be available although the urgency for its use is not great.

Recommended Steps in the Management of Acute Cardiac Arrest

ARTIFICIAL RESPIRATION

One-hundred per cent oxygen should be introduced into the lungs, and this is best accomplished by passing a tube into the trachea and maintaining an intermittent rhythm by compressing the rubber breathing bag or by a suitable mechanical device. In this way, it is possible to aid pulmonary blood flow and maintain adequate oxygenation of the blood which does reach the lungs.

CARDIAC MASSAGE

This is the single, most important step. It may be carried out through: (A) a transthoracic, (B) a transperitoneal subdiaphragmatic, or (C) a transperitoneal transdiaphragmatic approach. Cardiac massage may be carried out far more efficiently through the transthoracic approach however. The chest is entered through the fourth or fifth interspace. The costal cartilages above and below may be divided if necessary. It also has been found that the efficiency of massage is increased if the heart is grasped by the hand with the thumb in front and the fingers behind, rather than pressing the heart against the anterior chest wall. This massage should be regular and rhythmical. The compression should be gradual; the release, rapid. The cardiac output increases directly with the rate of massage—60 to 80 is considered an adequate rate. A rhythm more rapid than this requires a two-man team because it is too tiring for one and, therefore, may be impractical.

DRUG THERAPY

A. Adrenalin is a most valuable agent in reestablishing the heart beat. It increases the excitability of the myocardium and all surgeons who have employed it can attest its power. Over-dosage is frequent, however, and repeated small doses are vastly superior to one large one. One-tenth to one-third of a milliliter of adrenalin (1 to 10,000) may be given. This may be repeated in two to three minutes. Preferably, this injection is made into the right ventricular cavity.

B. Procaine is used to decrease the irritability of cardiac muscle and the conducting system of the heart. It is helpful in preventing fibrillation or if

ventricular fibrillation already exists, often it will restore the rhythm to normal. Injection of 5 to 10 milliliters of a 1 per cent solution is considered adequate dosage. Joseph et al.[11] in reporting their studies on pronestyl (procaine amide) found that when administered prophylactically, it decreased the incidence of all types of cardiac arrhythmias; and when given therapeutically, it reverted ventrical arrhythmias to normal sinus rhythm.

C. Calcium Chloride greatly improves myocardial tone and increases the strength of cardiac contraction. The effect appears to be a direct one on the myocardium increasing its excitability and contractility. Kay and Blalock[12] recommend that this injection be made into the left ventricular cavity so that massage can force the solution directly into the coronary arteries. Two to four milliliters of a 10 per cent solution is the usual dose and this may be repeated in two to three minutes if necessary. Cardiac massage should be continued during this time.

ADDITIONAL MEASURES

A. A ten degree Trendelenberg position aids the flow of blood to vital centers.

B. Intravenous fluids including blood and plasma increase blood volume and cardiac output.

Once artificial respiration and cardiac massage have been completed the urgency of the situation has ceased and time is no longer the prime factor. The patient can be maintained indefinitely without fear of increased brain damage.

Ventricular fibrillation with its rapid, uncoordinated, and ineffectual beats should be contrasted with cardiac arrest where there is complete absence of myocardial activity. Often these cannot be differentiated clinically and it may be impossible even when the heart is exposed. Nevertheless, the initial treatment is the same regardless of which condition exists. When ventricular fibrillation rather than complete arrest exists, normal rhythm may be restored after steps 1 and 2 have been completed. Procaine with its ability to desensitize the irritable myocardium should be administered as soon as myocardial ischemia is overcome with the massage. Frequently this is all that is required to return the rhythm to normal.

C. Electrical Shock is utilized to defibrillate the heart and restore a normal rhythm. Johnson et al.[13] explain that: "The purpose of applying an electric shock to the fibrillating ventricles is to produce a strong, simultaneous contraction of all the muscle fibers. This is followed by simultaneous relaxation and after a brief pause, regular rhythm is usually resumed." By placing electrodes on the myocardium, a charge of 110 volts and 1.5 amperes or approximately 130 volts may be passed through the heart, thus stopping the ineffective, twitching beats. Serial electrical shocks thrown in rapid succession may be necessary to accomplish this. Once fibrillation has

been converted to complete arrest, massage usually will restart cardiac activity

Recently, Callahagn and Bigelow[14] described an electrical pacemaker which has been used successfully in experimental animals to control heart action during cardiac standstill. The electrical impulses may be applied externally at thoracotomy or internally by introducing an electrode through the superior vena cava. This appears to be a relatively safe procedure and its possible clinical application will be awaited with great interest.

SUMMARY

1. Cardiac arrest, in its early stages, is a reversible condition.

2. Most cases can be saved if the condition is recognized immediately and therapy is instituted without delay.

3. The emphasis should be placed on prevention rather than on treatment.

4. The etiology and recommended treatment of cardiac arrest are discussed.

5. Surgeons and anesthetists should have a greater awareness regarding the existence of this condition.

6. The surgeon is open to criticism if he fails to institute vigorous resuscitative measures.

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CLINICO-PATHOLOGICAL CONFERENCE

Directed by: E. Clarence Rice, M.D.

Assisted by: Sanford L. Leikin, M.D.
Richard J. Waters, M.D.

By Invitation: Adrian Recinos, Jr., M.D.

Sanford L. Leikin, M.D.

This nine week old colored female infant was admitted to the hospital on November 1, 1950 and died on December 5, 1950. The patient was born at Gallinger Hospital on September 27, 1950. The delivery was normal and spontaneous. The birth weight was six pounds and twelve ounces. After remaining in the hospital for five days, the patient was discharged. She did well until three weeks prior to admission on a formula of evaporated milk, boiled water, and Karo syrup. At that time the infant developed a "head cold", and started to cough and vomit, the latter occurring after a bout of coughing. Her bowel movements were reported as normal. The family history was negative.

On admission the patient weighed five pounds and eight ounces. She appeared marasmic and extremely dehydrated. Her fontanel was sunken. There was generalized shotty adenopathy. The chest was clear to percussion and auscultation. No masses were palpable in the abdomen and the liver and spleen could not be felt. The neurological examination was negative.

Examination of the blood on the day of admission showed the hemoglobin to be 11 grams and the red blood cell count to be 3.7 million. There were 19,900 white blood cells per cubic millimeters of which 26 per cent were lymphocytes, 51 per cent neutrophiles, 19 per cent bands, 2 per cent monocytes and 2 per cent eosinophiles. The carbon dioxide combining power of the blood plasma was 42 volumes per cent. Urinalysis was normal. Examination of the spinal fluid revealed no abnormalities. Roentgen examination of the chest (Fig. 1) revealed an infiltrative lesion involving the medial third of both lower lobes, which was interpreted as a bilateral bronchopneumonia.

The patient was given parenteral fluids, and 100,000 units of aqueous penicillin was given every six hours. Some improvement was noted after the fluids were given. The patient took only 1 to 1½ ounces of milk at each feeding and had to be maintained on supplemental amounts of parenteral fluid. The infant regurgitated frequently but no evidence of abnormality of the esophagus was seen on x-ray examination using barium as a contrast medium. Endoscopic examination of the pharynx revealed no evidence of abnormality. Gavage feedings were given every four hours, and the patient retained about two ounces of each feeding. Because of cyanosis and inability to maintain a normal temperature the patient was placed in an incubator and given oxygen. Adrenalin eosinophile response tests were done on November 25 and November 30. The first showed a control level of 28 eosinophils per cubic millimeter which then rose to 92 four hours after the adrenalin. The tests done on November 30 showed a control level of 15 eosinophils per cubic millimeter with a drop to 10 after two hours and 10 eosinophils per cubic millimeter after 4 hours. The serum sodium was 299 mgms. per 100 milliliters, while the chlorides were 468 mgms. per 100 milliliters. On December 2, intramuscular injection of desoxycorticosterone acetate was started. Within the next two days, the patient seemed improved. She was taking 3 ounces of formula without vomiting. A re-examination of the chest revealed the pulmonary lesion to

have undergone considerable resolution, but some density in the upper right lobe was still present. On December 5 at about 11:30 A. M., the patient's respirations became irregular and of abdominal type. She was digitalized with 0.7 milliliters of digifolin intravenously and 25 milliliters of Darrow's solution were also given intravenously. The heart sounds remained distant, but were slightly improved following the administration of digifolin. At 2:15 P. M. on December 5, 1950, the thirty-fifth day of hospitalization, the patient expired.

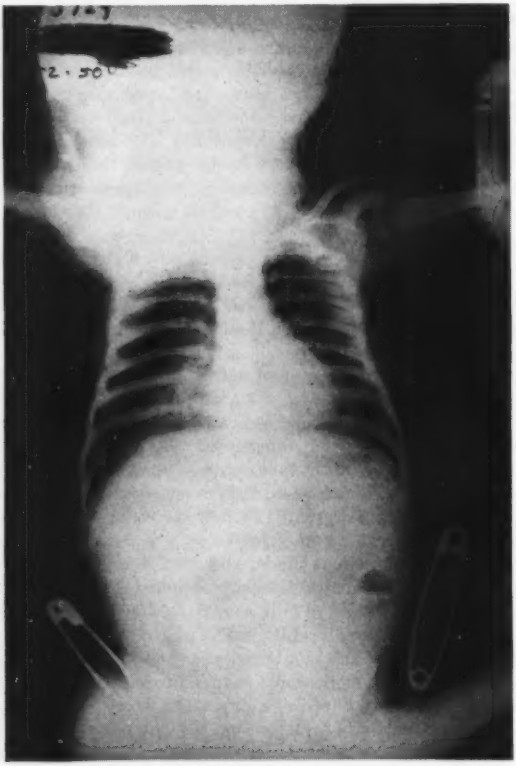

FIG. 1. X-ray of the chest on admission

DISCUSSION

Adrian Recinos, Jr., M.D.

We have for discussion the case of a colored female infant who was hospitalized at the age of five weeks with a history of cough and vomiting, signs of malnutrition and dehydration, a leucocytosis and granulocytosis,

and x-ray evidence of bronchopneumonia. She failed to respond appreciably to penicillin and oral and parenteral feedings, vomited frequently, became cyanotic and was unable to maintain a normal temperature. An incubator and oxygen were resorted to and several diagnostic studies, aimed chiefly at the persistent vomiting, were instituted. The child went steadily downhill and died five weeks after her admission to the hospital.

This problem may be approached from two directions. First, is the fatal process due entirely to a complication of the initial respiratory disease or, second, is there another obscure pathologic entity accounting for the persistent vomiting, malnutrition, and apathy?

At this age a common complication of pneumonia is atelectasis. This is brought about by the frailty of the rib cage and the inadequacies of the cough and swallowing mechanisms with the result that mucus is not properly expelled from the bronchi and ingested liquids may be aspirated into the lungs. An x-ray of the chest taken the day before the child died (fig. 2) is reported to show "considerable resolution" although some density remained in the upper right lobe. In addition, it should be pointed out that the interspaces are smaller on the right side than on the left, and there is another discrete density fanning out from the mediastinum in the lower right lobe which may represent an atelectatic process.

The other common complications of pneumonia, i.e. pleurisy with effusion, empyema and abscess can rather definitely be ruled out by the x-rays. Another complication of pneumonia which may terminate a patient's life is a bacteremia or septicemia with septic emboli capable of producing extensive infection of the brain, meninges, bones, joints, peritoneum and elsewhere. Although there is no evidence in the protocol that these existed, their presence cannot be ruled out.

It is possible, but not likely, that this patient had an unusual type of pneumonia which would not be expected to respond to penicillin. In this category would be tuberculosis, syphilis (pneumonia alba), lipoid pneumonia, and inflammatory processes due to Hemophilus and Friedlander's bacilli and other uncommon organisms.

An investigation of unexplained vomiting must include a study of several systems of the body. The esophagus and the gastro-intestinal tract cannot be indicted in this case. Contrast roentgenographic studies were done from both directions and no abnormality was noted in an endoscopic examination from above. There were no physical signs or x-ray evidence of a diaphragmatic hernia or pyloric stenosis.

Vomiting is a common manifestation of increased intracranial pressure whether due to an inflammatory, neoplastic or hemorrhagic process. Meningitis can be ruled out here by the normal spinal puncture. A tumor or an abscess would be an extreme rarity at this age. More plausible would be

the existence of an intracranial hemorrhage, particularly a subdural hematoma, which not infrequently first manifests itself by nonspecific symptoms of failure to gain weight and vomiting. However, some degree of hydrocephalus would be expected to have developed in the first ten weeks of life in an infant with a subdural hematoma.

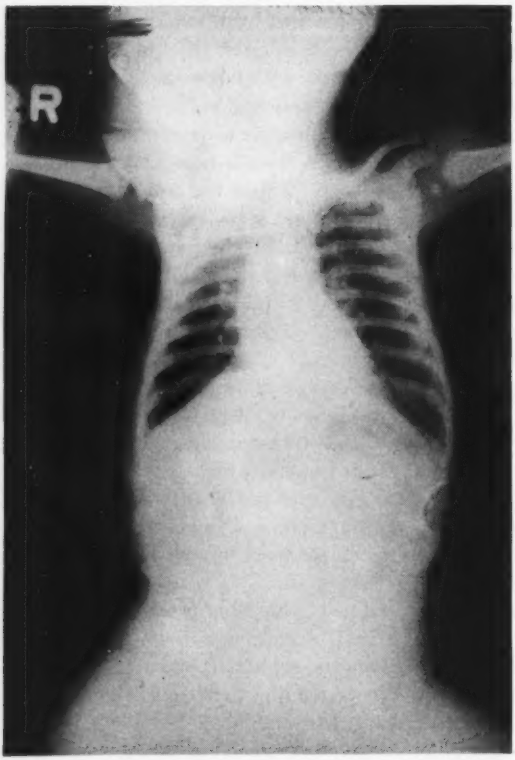

FIG. 2. X-ray of the chest on the day before death

The genitourinary system must always be considered in the differential diagnosis of vomiting. Malformations of this system are reported to exist in 5–12 per cent of all necropsies. In the first weeks of life the most likely lesions are polycystic kidneys or congenital hydronephrosis due to a stricture somewhere along the urinary tract from the pelvis to the urethra. The retention of nitrogenous excretory products in these conditions causes vomiting. The normal urinalysis reported in this patient tends to minimize but does not rule out an obstructive lesion of the urinary system.

Most important of the endocrine disturbances causing vomiting in infancy is adrenal cortical insufficiency. The diagnosis is made by demonstrating low concentrations of sodium and chloride in the serum and an increase in the potassium and non-protein nitrogen. The diagnosis is further established by the improvement caused by the administration of salt and desoxycorticosterone. Neither of these criteria are fulfilled in our patient. The sodium and chloride values do not fit the picture and there was no improvement with desoxycorticosterone.

In conclusion, I can only reiterate the entities which seem the most likely causes of this infant's illness and death. These are:

1. Atelectasis and aspiration pneumonia
2. Subdural hematoma
3. Congenital hydronephrosis or polycystic kidneys
4. Unusual type of pneumonia.

PATHOLOGIC DISCUSSION

Sanford L. Leikin, M.D.

The body was that of a poorly developed, poorly nourished, colored female infant whose weight was 2.6 kilograms and length was 67 centimeters. The head circumference was 34 centimeters and the chest circumference was 29 centimeters. There was 5 milliliters of yellow, clear fluid in each pleural cavity.

The respiratory system demonstrated the major pathology. The lumen of the lower trachea and bronchi contained a large amount of thick, yellow, muco-purulent material. The left lower lobe and the medial portion of the right lower lobe were light blue and firm. The lungs cut with ease and on cut section the surfaces of the above described areas were somewhat dry and mottled yellow. A small amount of blood, yellow frothy fluid and thick purulent material exuded from the cut surface on manual pressure. The remaining portions of both lungs were pink and crepitant.

On microscopic examination, the alveoli of the lungs were packed with polymorphonuclear leukocytes, lymphocytes, macrophages, and pink staining fibrin. The capillaries of the alveolar walls were congested. There was moderate peribronchial infiltration with lymphocytes, polymorphonuclear leukocytes and a few macrophages. The bronchi showed exfoliation and separation of their lining cells.

The liver shows some evidence of engorgement with vacuolization of the cells. The remaining organs showed no significant gross or microscopic pathologic changes.

The pathologic diagnoses are:

1. Lobar pneumonia
2. Acute hepatic congestion.

The gross and microscopic examinations of the organs of this patient demonstrate that the primary disease was representative of an acute inflammatory process involving the lungs. The gastro-intestinal symptoms and malnutrition which this infant manifested were probably due to the respiratory infection. However, no anomalies of the upper respiratory system or the gastro-intestinal tract were present. Antibiotics were used but they did not seem to have a profound effect. It is possible that the organism present was penicillin-resistant although a culture of bronchial secretions at autopsy revealed only what was probably a contaminant.

The cause of death in this patient is obscure. It is possible that hypokalemia was produced by the use of desoxycorticosterone acetate, but this cannot be definitely determined as no electrocardiogram or potassium level was obtained prior to death.